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## Necrohemorrhagic enterocolitis caused by *Clostridium perfringens* type C in a foal

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**O**n May 31, 1989, a seven-day-old Belgian colt was found late at night with signs of colic. A clinician from the Faculté de médecine vétérinaire de l'Université de Montréal was called to the premises. Upon his arrival the foal was recumbent, weak, and moderately dehydrated with a rectal temperature of 38.0°C and a heart rate of 110/min. It had signs of severe abdominal pain; however, diarrhea was not observed. The foal died a few minutes after intravenous fluid therapy was initiated. From birth until the morning of the day it died, the colt had appeared healthy and was kept in a large box stall with its eight-year-old primiparous dam. The stable was clean, well-ventilated, and it sheltered five other adult horses of various breeds.

The foal was necropsied approximately nine hours after death. External examination of the carcass revealed the presence of darkly-stained and fluid fecal material in the perianal area. The abdominal cavity contained approximately 0.5 L of serosanguineous fluid. A severe and extensive hemorrhagic enteropathy involved nearly the whole length of the small intestine, with the exception of the proximal part of the duodenum. The cecum and portions of the large colon were similarly affected. The serosal surface of affected intestine was hemorrhagic and edematous, and the mucosa was yellow or brown and necrotic (Figure 1). The intestinal content was fluid, red-brown, and intermixed with necrotic debris. Mesenteric lymph nodes were swollen and congested. Synovial fluid from all joints was tinged with blood. Other tissues and organs appeared normal.

Significant histological changes were restricted to the intestinal tract. The wall of the small intestine, particularly the submucosa, was markedly hyperemic, edematous, and focally hemorrhagic. There was diffuse, mild to moderate infiltration of the submucosa with neutrophils and some macrophages and plasma cells. Fibrinous thrombi were present within the lumen of several submucosal and a few mucosal blood vessels. In most sections there was massive coagula-



Figure 1. Segments of partially opened jejunum showing hemorrhagic serosa and necrotic mucosa.

tion necrosis of the entire mucosa. The necrotic mucosa and occasionally the submucosa of these severely affected intestinal segments were invaded by a heavy, heterogeneous bacterial flora. However, in a few sections the necrosis was mostly restricted to the villi. Affected villi were devoid of surface epithelium and were covered with gram-positive bacterial rods. Similar lesions were found in the cecum and large colon.

On bacteriological examination, the spleen and mesenteric lymph nodes yielded *Escherichia coli* and alpha-hemolytic streptococci. Culture of the liver was negative. Gram-stained smears of duodenal, jejunal, and ileal mucosal segments revealed the presence of numerous gram-positive rods. Aerobic cultures of intestinal segments yielded *E. coli*. *Clostridium perfringens* was isolated in large numbers from anaerobic culture of these specimens and the isolate produced its toxin in a cooked-meat medium (Difco, Detroit, Michigan, USA). The identification of the toxin was carried out by the neutralization test in mice, using *C. perfringens* type-specific diagnostic antisera against types A and C (Wellcome Diagnostics, Dartford, England). Tests were conducted according to the manufacturer's instructions and the toxin-neutralization pattern was consistent with that of a type C strain.

*Clostridium perfringens* type C enterotoxemia is an uncommon disease of worldwide distribution charac-

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terized by an often fatal hemorrhagic and necrotizing enteritis affecting newborn animals of several species including calves, lambs, piglets, and foals (1). In Canada, the disease in foals has been reported only once from the West (2); in eastern Canada, it has been reported only in piglets (3).

Presumptive diagnosis of *C. perfringens* enterotoxemia is usually based on necropsy findings and the presence of large numbers of gram-positive rods in intestinal smears. However, it has recently been shown that similar findings in foals could be attributed to *C. difficile* (4). The search for *C. difficile* in the specimens from the present case was unsuccessful, and *C. perfringens* type C was considered the cause of the severe enteropathy. Definitive diagnosis is obtained by demonstration of *C. perfringens* type C beta toxin in

the intestinal contents or from the isolate. Immunization of dams against the organism appears to be the most practical and effective approach to prevention of this disease in the newborn in enzootic areas (1). CVJ

## References

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3. Morin M, Phaneuf JB, Malo R. *Clostridium perfringens* type C enteritis in a Quebec swine herd (Letter). Can Vet J 1981; 22: 58.
4. Jones RL, Adney WS, Alexander AF, Shideler RK, Traub-Dargatz JL. Hemorrhagic necrotizing enterocolitis associated with *Clostridium difficile* infection in four foals. J Am Vet Med Assoc 1988; 193: 76-79.

## Thailand Anyone?

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The 7th Federation of Asian Veterinary Association Congress takes place November 2-4, 1990 in Thailand. This large international meeting is conducted in English and covers a wide range of topics.

If a number of CVMA members are interested in attending this meeting, the CVMA could develop an official presence at the meeting. As the CVMA and the Agency for International Veterinary Development are working towards creating short work periods overseas for Canadian veterinarians, this opportunity could serve as a very important meeting to form linkages with South East Asian colleagues.

In addition, group tours to Singapore, Hong Kong, veterinary colleges and farms could be organized to enhance your visit.

Interested individuals should contact:

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